Hyponatremia : pathophysiology, treatment and prognostic value Summary of the discussion

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Clinical consequences of chronic hyponatremia

Hyponatremia in cirrhosis, like in other diseases, has been generally considered as a minor clinical event without real repercussion on the physical and mental status of the patient. However, it has been demonstrated that mild hyponatremia (> 125 mEq/L) and even very mild hyponatremia (serum sodium level 127-132 mEq/L) is associated with an increased risk of falls, probably due to impairment of attention, posture and gait mechanisms (1). It was noted that in volunteers of similar age, after mild alcohol intake (0.55 g/kg body weight), attention and gait tests were less affected (1). Patients with severe hyponatremia should be advised not to drive. Hyponatremia is also an independent prognosis factor in case of cirrhosis as pointed out by several recent studies and might be added to the MELD score in order to improve organ allocation in patients on the waiting list for liver transplantation.

Evaluation of renal function in decompensated cirrhosis

The best test is the glomerular filtration rate assessed by isotopic method (2). However, this test is not very used in clinical practice. The performance of the Cockcroft and Modification of Diet in Renal Disease study (MDRD) equations are poor in case of decompensated cirrhosis. Furthermore, measurement of creatinine may depend on lab methodology and physiological factors such as age, sex, nutrition status or bilirubin level.

Therapy of dilution hyponatremia

Water restriction remains one of the pillars of the treatment in case of dilution hyponatremia. It is important to educate the patient and ask him to note consumption of liquid. For example, it is common that the patient does not take into account the consumption of coffee.

Administration of hypertonic salt is no appropriate therapeutic option. Indeed, giving salt will result in water retention. Nevertheless, there is a clinical setting where giving sodium is a required emergency. It is when hyponatremia is severe, acute and results in epilepsy. This clinical setting is not generally observed in cirrhotic patients but may be observed in mentally disturbed patients suffering from potomania. In these patients, severe and acute hyponatremia may lead to cerebral oedema and respiratory arrest occurring around 30 minutes after the seizure episode.

Administration of salt and furosemide is an easy tool insufficiently used in clinical practice : the sodium load is preserved while water retention is avoided. Nevertheless, it is a somewhat dangerous tool and this therapeutic option should be used only in hospitalised patients under strict surveillance.

Administration of lactulose has a mild corrective effect on hyponatremia.

V_2 receptor anatagonists are a true advance in the treatment of dilution hyponatremia. The safety of these drugs is good, without reported severe side effects. Particularly, these drugs do not result in an activation of the neurohumoral agents renin, angiotensin, aldosterone or catecholamines.

Administration of -opioid agonists is efficient in the treatment of dilution hyponatremia. Nevertheless, the development of niravolin has been stopped because of side effects, in particular respiratory failure.

References

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